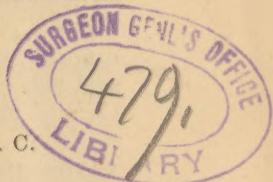


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When Young Simons did
Read before the S. C. Med. Soc.
June 1891

ACUTE INFECTIOUS HÆMOGLOBINÆMIA OF THE
NEWLY BORN.

BY T. GRANGE SIMONS, M. D., CHARLESTON, S. C.



Mrs. ——, a healthy primipara was delivered of a vigorous female infant on the 10th May, '91, at 4:30 A. M. The rupture of the membranes without pain was followed by three or four strong expulsive pains, and the infant was born before the arrival of the nurse or myself. The perineum was deeply torn and was immediately repaired with deep silver wire sutures and dressed with Iodoform. The infant cried strongly and all seemed well, urination and defecation from the infant attracted no attention as in any way being abnormal. On the night of the 12th and morning of 13th, inst the child was restless and declined to nurse, bowels confined, abdomen distended somewhat. Ol. olivae was given and two dark mucous discharges, accompanied with tenesmus occurred, a carminative of

R.—Tinct. Assafetid. gtt. viii.

Tinet. Lavandul. Comp. . . m x.

Spts. Ammon. Arom. . . m. viii.

Sod. Bi-Carb. grs xx.

Aq. Anis. Ft. 3 i.

M. S.—Take a tea-spoonful every hour or two.

The child seemed in a state of apathy—it would not nurse nor cry, it would take the mother's milk from a spoon and no trouble in swallowing existed. The discharges from the bowels dark, of a tarry hue, with great tenesmus. At 11.30 A. M., May 13th, the child became cyanosed, cold extremities, convulsive or choreic twitchings of face and limbs, especially marked on the left side. Thinking I had a case with congenital cardiac disturbance, directed the right postural position on a pillow with dry warmth. 4.30 P. M. the discharges from the bowels of dark mucus, respiration and pulsation not materially affected, temperature normal, skin moist, hot, suffused with a dark pink color, the convulsive

Presented by the author

movements constant. I now directed Bromide of Soda gr. ii to be given every two or three hours, with the carminative already prescribed. This seemed to influence the convulsive movements, yet the facial contortions were still apparent at times. The jaws were not contracted, nor closed in a tetanic manner, the tongue appeared to be too large for the mouth and was often protruded in a restless, uncomfortable way. Careful examination failed to detect any lesion or injury to neck or spinal column, a slight glairy discharge from vagina and rectum was observed. The condition of the child impressed you as one of great discomfort rather than of danger. The apathy or inability to nurse or cry was very evident. It continued to take its nourishment from the spoon and was well supported and continued to grow. Early on the night of the 14th May, the urine became dark and apparently bloody. The dusky hue of the skin became of a tawny or icteric tinge, the conjunctivæ showing the icteric condition most markedly, the fæces also became more dark, tarry and mucoid, the choreic movements were still observed but not intensified.

The urine under the microscope showed the presence of dark granular masses, no blood corpuscles, nor tube casts, albumen in small quantity, or a condition of Haemoglobinæmia. The diagnosis was now made as "Acute Haemoglobinæmia," or Winkel's disease.

Tinct. Ferri Chloridi gts. iii. in water was directed to be given every six hours, the child to be kept quiet, warm and free from disturbance, noise and exposure to the bright light (electric) as its eyes were very sensitive to such influences. The Haemoglobinuria continued for two days, then the urine became occasionally clear, but this condition fluctuated for three or four days longer. The convulsive movements ceased. The apathy and inability to cry continued, nor would she nurse; would allow the nipple to remain in her mouth, but could not make traction upon it. On May 16th, the sixth day since her birth, the face, neck, legs and arms showed an eruption of vesicles or bullæ of various sizes, some sudaminal, others quarter of an inch in diameter, all filled with a turbid serum, the lips and eyelids swollen from the presence of the eruption, no areolæ, or inflamed base surrounded the vesicles. Warm baths with borax dissolved in them were now employed and

R.—Ungt. Zinc. Oxid.

Ungt. Aq. Ros. à à ʒ ss.

The vesicles dried up in 36 to 48 hours, but a crop of smaller size succeeded them, some of the larger bullæ were punctured at their bases, and the serous fluid allowed to exude. After desication of the bullæ there were left dark pink spots covered with branny scales, but no ulcerative process at any time was shown. The mixture with the Bromide of Soda was discontinued when the cutaneous lesions were shown. The Tinct. Ferri Chloridi was still continued, with an increase of one drop, or four drops every 6 hours. The urine was now normal, and the fecal discharges natural as to consistency, but showing the color from the iron. The child cried and nursed, but not with avidity, the tongue seemed to embarrass it and was still frequently thrust out between the lips as if in the way when the nipple came in contact with it. Such is the clinical history of the case.

The literature of this disease is not voluminous, nor is the etiology or pathology clearly defined. The best article that I have had access to is by J. P. Crozer Griffiths, of Philadelphia, in *Encyclopedia of Diseases of Children*, Keating, vol. iii. I shall freely use this article. The varied synonyms collected by him nearly all have allied nomenclature. It is needless to repeat them. The definition given is: "An acute and usually epidemic affection of the blood, developing in the new born, characterized by cyanosis, icterus and hæmoglobinuria running a rapid course."

Griffith states that in 1871, Pollack, of Vienna, first reported 12 sporadic cases. In 1873, Larroyenne described an epidemic in the Lyons Maternity Hospital. Bigelow reported in the Boston Lying-in Hospital 10 cases, and published his cases in the Boston Medical and Surgical Journal, March 11th, 1875. In 1877, Herz reported two cases of acute fatty degeneration of the new born that appeared to be identical with the disease now developed. Winkel, in 1879, described more fully the history of twenty-four cases in the Dresden Maternity Hospital, these cases occurred within one month. He called it "a hitherto undescribed endemic disease among the new-born. From this account his name has been linked with the disease, Birsch-Hirschfield made some publication about this period with a pathological report of autopsies of these cases. Some few other cases have been described by other observers.

The Etiology is not well understood. In the case now reported, all sanitary conditions were good, so as to exempt the mother from any source of danger or discomfort. At

first the rapid delivery was adjudged a factor that might have produced profound centric disturbance, and that a case of "Trismus" was to be dealt with. Toxic influences could hardly have had any effect as to cause. Creolin was used in the vaginal douche employed for the mother, and the torn perineum was dusted with iodoform, but I never omitted the use of either agent, so little did I regard them as a source of danger to the infant.

Vaso-motor disturbances produced by cold or heat, as causing haemoglobinæmia is adduced by Ponfick and others. The child was born at 4.30 A. M., on a mild May morning, and all care was used by a competent, trained nurse in dressing it. Tyson on Haemoglobinuria, page 526, Vol. I, Annals of Medical Science, 1888, refers to this condition occurring in malarial subjects, especially after severe muscular exercise, followed by sudden chilling of the body. Bunkerstein also alludes to like causes, especially when much gastric disturbances occur. When we have the malarial dyscrasia an additional factor presents itself in producing haemoglobinæmia, although Baker, of North Carolina, thinks that another additional factor exists in the so-called malarial haemoglobinuria besides the malarial miasm, and regards the destruction of the red blood corpuscles as due to a special bacillus.

Silbermann, as quoted by Guiteras, injected dogs with substances causing a solution of haemoglobin. This produced venous plethora, with obstruction of the veins by deposits, dyspnoea, cyanosis, reflex irritability, high temperature, convulsions and haemoglobinuria, followed by death.

Hæslin, Munich Medical Wochenschrift, 1890, p. 248, American Journal Medical Science, August, 1890, in discussing the fact of the diminution of haemoglobin in anæmia and chlorosis, believes the loss is not due to destruction of the red blood corpuscles in the vessels, but simply to hemorrhage, especially concealed gastric hemorrhages, and claims that autopsies have shown conclusively evidences of this in cases with anæmia. The icteric hue of the skin may be due to the re-absorption of haemoglobin from the gastric or intestinal ecchymoses, as well as by its formation or destruction in the blood vessels. The presence of iron and haematin was much less in the stools as the anæmia decreased.

Sherenziss, as quoted by Griffith, states that the blood of the new-born is of lower specific gravity as it is deficient

in hæmoglobin. Kruger also agrees with this statement. Leichtenstern claims that the hæmoglobin is greater at birth, or soon after, and least from the first to the fifth year, when the increase is again noted, the decrease of hæmoglobin was observed within two weeks after birth.

Silbermann states that Hæmoglobinæmia is physiologically present in the new-born, as shown by the bile in the urine and the peculiar shadows in the blood under the microscope.

The cause of Hæmoglobinæmia is the presence of an agent circulating in the blood that may produce a destruction of the red blood cells. Hydræmia is the condition of the new born, and cold or other depressing influences may tend to produce or intensify the changes in the blood cells.

Hæmoglobinæmia is a condition of the blood in which Hæmoglobin is circulating free in the plasma, nor is Hæmoglobinæmia always accompanied by Hæmoglobinuria.

Ponfick has shown that the liver converts Hæmoglobin into bile pigments. Dr. Ralfe, in a paper read at Brighton, before the British Medical Association *Lancet*, Oct. 23, '86, shows that the blood crystals or coloring matter may not appear in the urine, but is converted by the liver into urea and urobilin, both of which may be found in the urine to excess, that from vaso-motor disturbances, we may have destruction of the blood corpuscles. Hence the value of Iron and Quinine in such cases, as they diminish nervous irritability. He claims in many disturbing states of the system, the red blood corpuscles may undergo disintegration in the liver, the coloring matter of the hæmoglobin is changed into bilirubin, subsequently into urobilin, whilst the remainder of the protoplasm of the blood cells is converted into urea. The anæmic condition that ensues is owing to the decrease of the red blood corpuscles. The excretion of urea was small, but when the anæmia was relieved, the excretion of urea increased.

The question of diagnosis between the morbid conditions that simulate Acute Hæmoglobinæmia, may, at first, give us some difficulty. The doubt may arise as to its identity with the "Acute Fatty Degeneration of the New Born," or "Buhl's Disease." I quote freely from Griffith here, not following his text, but the opinion given: Much similarity may appear, but in "Buhl's Disease," the infant is usually born asphyxiated, and no appreciable cause exists for this condition. They rapidly die or show symptoms of fatty degeneration; nor is Hæmoglobinuria found in Buhl's Disease. Hæmorrhages

from stomach, bowels, umbilicus, or extravasations of blood under the skin, or mucous surfaces, and a more chronic course differentiate it from Acute Infectious Hæmoglobinæmia, from poisoning by Chlorate of Potash, Carbolic Acid or Phosphorous. The absence of these agents must be considered. True, in Chlorate Potash poisoning, we have cyanosis, icterus and the urine also shows the presence of Hæmoglobin or Methæmoglobin and brownish casts with remains of the red blood corpuscles, the blood is of a chocolate brown, with many colorless corpuscles. The tubules of the kidneys are filled with casts, echymosis under the mucous membranes, dyspnoea, vomiting and diarrhoea also occur with Chlorate Potash poisoning. The liver also, after death, shows an enlargement, with brown detritus, resulting from the disintegrated blood corpuscles. The dark or blackish green urine of Carbolic acid poisoning when treated with Nitric acid, then with Potash, shows a reddish, then a pea-green changing to a violet hue; without this chromatic test being shown Carbolic acid is not present. Cyanosis from congenital malformation of the heart, would not show the other symptoms noted.

Prognosis unfavorable. Of the 71 cases reported in Griffith's article only 5 are recorded as having recovered. Winkel reports 24 cases, with 1 recovery; Bigelow 10 cases, with 2 recoveries; Pollack 12 cases and 2 recoveries.

The duration of the cases variable, but all short. The fatal cases of Pollack died on the second or third day. Bigelow's cases averaged five days. Winkel's averaged 22 hours, the shortest 9 hours, the longest four and a half days. Sander's case 6 hours. Strellitz's case two days. Herz one or two days. Most of Winkel's cases died in convulsions or collapse.

Treatment.—In cases of such short duration, often no treatment will avail. In the case reported, carminatives with alkalies were given with a view of relieving the gastric and intestinal or colicky symptoms. Constipation was relieved by oil and also, glycerine enemata. For the convulsive disturbance when it became apparent, Bromide of Soda, gr. ii, was given with the carminative mixture every two or three hours; as soon as the exact nature of the case was shown by the icteric condition and the Hæmoglobinuria, Tinct. Ferri Chloridi, gtt. iii, in water, every six hours, in addition to the Bromide treatment. The Bromide was omitted, as soon as the convulsions ceased, and especially as the skin lesions became also evident.

The administration of Tinct. Ferri Chloridi was carried out in Bigelow's favorable cases. I regarded it beneficial not only for its hæmostatic properties, and for the purpose of diminishing the loss of Hæmoglobin, but also as aiding its formation, and for its influence, upon the vaso-motor system, as it doubtless, allays or averts morbid changes set up by the irritability of the nervous system. Quinia may have been indicated in an older subject, but I did not care to risk gastric irritation, which Quinine so often produces in children. Arsenic was theoretically indicated to give stability to the red blood corpuscles, it was not, however, administered, as the case improved. The child was kept warm and quiet on a pillow. Mother's milk was given regularly from a spoon until the power of nursing was restored. The cutaneous lesions were treated with warm baths, in which Borax was dissolved, these were used night and morning, and served to allay the inflammation materially. The larger bullæ were carefully punctured at their bases, with a fine needle and the contents being allowed to exude and were wiped off with absorbent cotton. Ungt. Zinc. Oxid. and Ungt. Aq. Ros. a a ȝ ss was also used as a dressing. The child convalesced and became vigorous.

18 Montague street, Charleston, S. C.
June 6th, '91.

